Address

ON

Vertigo of Bulbar Origin.

Delivered before the Harveian Society of London at the Annual Meeting, Jan. 16th, 1890.

BY

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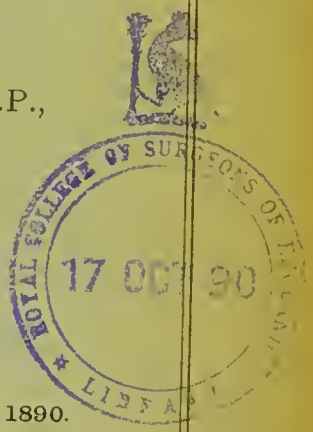

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ADDRESS ON VERTIGO OF BULBAR ORIGIN.

GENTLEMEN,—It is well known that vertigo is a symptom which may occur in connection with many conditions. With epilepsy, for example, in cases of oculo-motor paralysis, in disease of the cerebellum, in states of nervous exhaustion, or as a result of gastric disorder. We owe to Ménière our knowledge of the fact that vertigo of apoplectiform character, which was previously supposed to denote grave disease of the brain, may be dependent upon a strictly localised lesion of the labyrinth or internal ear. The bony structure of the internal ear is hollowed out, as you are aware, into three divisions, the vestibule in the centre, communicating in front with the cochlea, and behind with the semicircular canals, on the outer side with the tympanic cavity by a hole which is filled by the base of the stapes, and on the inner side with the internal auditory meatus. The membranous labyrinth lines more or less loosely this bony structure. Omitting details, it contains a fluid called the endolymph, and its walls support minute ramifications of the auditory nerve. In case of injury to the semicircular canals or their contents it is not the sense of hearing which is damaged, but a disturbance of equilibrium is occasioned. It is now, I suppose, very generally agreed that the function of the auditory nerve is twofold. By its distribution to the cochlea it is the nerve of hearing, and according as this portion of the nerve is stimulated or its structure damaged it will give rise to sensations of sound or to more or less hardness of hearing. By its distribution to the semicircular canals the movements requisite for preserving the equilibrium of the body are influenced. The functions of these two divisions, then,

are perfectly distinct. A disease of the bone, or of its membranous lining, will interfere with both or either of the functions, according to the extent to which the distribution of the auditory nerve fibres to both the cochlea and the semicircular canals, or to either alone, is thereby affected. It is evident, therefore, that as a result of disease in this region we may find more or less severe vertigo, accompanied, if the cochlea be affected, either by noises in the ear, or by greater or less deafness, and sometimes by both.

I am sorry to be obliged to detain you by reference to these points, but it is necessary for me to do so, in order that the line of my argument, if so it may be called, may be intelligible. Ménière's important exposition has extraordinarily influenced the diagnosis of vertigo. Paroxysms of vertigo, occurring in a person of previously fair health and accompanied by deafness or subjective noises, are now constantly being attributed to disease of the labyrinth. In the large majority of these cases the most careful examination discloses no evidence of disease of the outer or middle ear. Where some deafness exists, however, it is found that the tuning fork is heard very indistinctly or not at all when placed on the vertex, but better when held opposite the external meatus. These characters tend to show that the nerve of hearing is in itself in some way implicated in disease. In these circumstances it is customary to suggest that inflammation, hæmorrhage, or effusion has taken place into the labyrinth. Should, however, there be evidence of disease in the middle ear, or obstruction of the Eustachian tube, or irritation or obstruction of the external auditory meatus, it is concluded that the labyrinth is indirectly affected. In either alternative, then, the immediate cause of the vertigo is ascribed to a lesion within the labyrinth.

Now, I have myself no doubt that paroxysmal vertigo, accompanied by deafness and singing in the ears, is sometimes occasioned in this way. Either a direct or indirect affection of the membranous or bony labyrinth causes irritation of the auditory nerve-endings and consequent disturbance of equilibrium, deafness and tinnitus. In all cases, indeed, in which vertigo is accompanied by these symptoms, there can be no reasonable doubt that to the auditory nerve we must in some way or other look for an explanation. My impression, however, has long been (and this is the point which I wish to bring before you to-night) that in a large

number—perhaps the majority of cases of auditory nerve vertigo—it is through its centre in the medulla oblongata that the nerve is affected, and not at its periphery. I cannot help thinking that the ear is very frequently indeed accredited unjustly with the production of this kind of vertigo, and that, if we could examine the whole complicated apparatus contained in the temporal bone, we should discover no disease whatever in it. The point, let it be understood, is this : The fact that disease of the labyrinth is apt to give rise to vertigo, accompanied by disordered function of hearing, has been practically construed, as it seems to me, into the idea that all vertigo accompanied by auditory symptoms depends upon a local affection of the labyrinth. I believe this to be an error, and would look to the medulla oblongata, in which the auditory nerve has its origin, for an explanation of the symptoms in a large number of cases of auditory nerve vertigo.

Of the ten cases published in Ménière's treatise in one only was there an autopsy. The case was that of a young woman who had been exposed to cold whilst menstruating. She suddenly became completely deaf, and after suffering from constant vertigo and vomiting died on the fifth day. On *post-mortem* examination nothing could be discovered in the nervous centres, but the semicircular canals in each ear contained a reddish plastic substance. There are a few—a very few—confirmatory cases of autopsy by subsequent observers. These cases, whilst to my mind quite sufficient to establish the fact that vertigo of the kind we are discussing can be occasioned by an irritative lesion of the labyrinth affecting the auditory nerve-endings, entirely fail to prove more than that this is one mode in which the auditory nerve may be affected in cases of this description. The inference that this is always the explanation of this kind of vertigo appears to me to be quite unwarranted by the circumstances.

If you will bear with me I will try to explain the grounds which appear to justify this opinion. The subject is an extremely difficult one, as must always be the case where we have to do with disorders of this class. As physicians, we are called upon to give relief in numerous cases of disorder of the nervous system in which there is no distinct evidence of organic disease. They are, indeed, those in which our aid is most frequently of all invoked. In calling them functional disorders we only imply that the disordered

function is what is observed, and of course the probable existence of an organic cause for the perverted action is not denied; indeed, I should rather say that this was taken for granted. The subject has an importance also beyond the mere interest attaching to the solution of a difficult problem, inasmuch as our means of treating a very common and distressing malady are on the whole likely to be useful in proportion to the success with which we master its pathology. Let me first refer to some examples, but I will make the accounts very short.

I was hurriedly sent for four years ago to see a medical man, forty-three years old, who had been lying seriously ill for two days. I found him in bed, and two medical friends who had been looking after him were with him. It appeared that two mornings previously he had woke up giddy and gone to sleep again. On waking again and getting up he fell down, looked extremely ill, and nearly fainted. He was got to bed, and there he remained for some weeks. So long as his head was kept perfectly quiet he was easy, but the slightest turn of the head brought on terrible giddiness. There was no deafness or tinnitus. He was away from work for many weeks, but returned well. He had been working again for some time when one day he was seized with faintness lasting a couple of hours, and accompanied by profuse sweating, and a similar attack occurred next day. In these attacks he did not lose consciousness, but felt as if his heart were not acting. Immediately afterwards he passed a large quantity of colourless urine. He described how in this attack, when he lay down and rested his head on the right cheek, he got an electric shock-like feeling and dizziness, but if he "eased off" his head by supporting it first with a pillow and gradually turning it round, he could avoid the giddiness. He could turn his head rapidly from side to side and stoop to pick up things, but he could not look up at an object to his left above him, because in that position the head is lowered to the right. The urine was acid, normal in quantity and character. There was no free uric acid. There was no evidence of organic disease of the heart. This patient had a gouty mother, but had not himself suffered from declared gout. The exacting character of his professional work, however, rendered him predisposed to goutiness.

It seems a little curious perhaps that after dwelling upon vertigo accompanied by deafness or noises in the ear I

should illustrate my remarks by quoting a case in which the hearing remained quite unaffected and there was no tinnitus. But the character of the vertigo in this instance so strongly points to its connection with the semicircular canals that we do not need the other symptoms in order to refer it to disorder in the district of the auditory nerve—in that section of it, at least, which is distributed to the semicircular canals. The faintness may be considered as probably due to inhibition of the heart's action through the vagus nerve. In his first attack the vertigo, which, it will be agreed, was typically characteristic of that referred to the auditory nerve, was the most prominent symptom. In the second attack it was the heart's function, probably through the vagus, which was most powerfully affected. This was accompanied by profuse sweating and large discharge of colourless urine, symptoms which may fairly be referred to irritation of centres situated in the medulla oblongata. The legitimate inference appears to be that the nucleus of the vagus, the auditory nucleus, as well as the vaso-motor centre in the bulb, were all subject to some irritating influence, which somewhat varied in the point most severely affected on the two occasions.

It has been frequently suggested as an explanation of the coincident troubles in the region of other nerves observed in cases of Ménière's vertigo that these are brought about reflexly through irritation of the ultimate branches of the auditory nerve distributed to the semicircular canals. But in the case just related it seems much more probable, as I have said, that the irritation was applied to the central ends of the nerves in whose district disorder of function was observed.

It is necessary to consider whether it is likely that irritation exerted in this situation would produce such disturbance of function, and for this purpose we may refer to the evidence afforded by other analogous conditions. Several years ago, when the subject of Ménière's vertigo was under discussion at one of the medical societies,* I suggested, as a possible explanation of the symptoms, that something like the nerve storm of migraine swept the medulla oblongata, and set up in the auditory nerve a condition which would give rise to neuralgia in a nerve of common sensation. I thought that the vertigo might pro-

* "Clinical Society's Transactions," 1876, p. 101.

bably be the expression of such an influence applied to the auditory nerve centre as would occasion pain if applied to the central origin of a nerve of common sensibility. What we call neuralgia, when it does not depend upon inflammation or some coarse irritation of the nerve trunk, is probably due to some change affecting the central origin of the nerve. Sensory nerves are excitable in their entire course from centre to periphery, and pain is produced by irritation of any part, although, according to the law of peripheral perception, it is always referred to the periphery. Supposing the change to affect the sensory nucleus of the trigeminal nerve in the medulla oblongata, we should expect to get pain referred to that portion of the distribution of the nerve which is represented in the particular section of the nucleus exposed to the irritating influence. The influence, then, of whatever nature it be, whether applied to the fibres of the fifth nerve or to its nucleus in the bulb, in either case gives origin to pain in the district of distribution of the nerve. But a like irritating influence applied either to the fibres or the nucleus of the auditory nerve will not of course give rise to *pain* referred to its distribution—for the auditory nerve is a nerve of special sensation—but to perturbed action in the organs supplied by the nerve, to tinnitus if the cochlear portion be subjected to the influence, and to giddiness if the part distributed to the semicircular canals be affected; and this whether the trunk of the nerve or its nuclear origin be the part attacked. This at least appears to me to be what must naturally be expected. It may be asked, granted the probability of irritation either of the trunk or of the nucleus of a nerve giving rise to a similar result, is there any kind of proof that it is so? I think there is.

It occurred to Professor Pierret of Lyons, in a necropsy of a case of tabes characterised by neuralgic pains in the district of the trigeminal nerve, to search for changes in the descending root of the fifth, which passes downwards to the lower part of the medulla oblongata. He found that there did exist around this inferior nucleus of the trigeminus a well-defined sclerosis, the form and connections of which recalled exactly what is seen in the posterior columns of the spinal cord in cases of tabes. There is a case of tabes now in hospital in which bulbar symptoms are marked and the patient suffers from violent pains in the district of both fifth nerves, due, there can be no doubt, to sclerosal irritation of the descending roots of the trigeminals.

In the year 1880, when bringing some cases of Charcot's joint disease before the Pathological Society, I offered the suggestion that the attacks of vomiting and pain in the stomach (gastric crises) occasionally observed in the course of tabes were probably caused by irritation of the roots of the vagus, and subsequently pointed out that a similar explanation would account for the occurrence of the so-called laryngeal crises. In all cases of tabes in which "lightning" pains have occurred during life, sclerosis of the root-entrance zone of the posterior column of the cord has been found after death, and this lesion may reasonably be looked upon as the proximate cause of the pains which, in accordance with a well-known law, are referred to the periphery. The resemblance between the gastric crises and the lightning pains is marked in the apparent spontaneousness of their occurrence and in the strange irregularity by which they are both characterised—now present in rapidly recurring paroxysms for hours or days, or even weeks and months together, and now entirely absent for varying periods of time. It appeared to me that if sclerosis, which when it involved nerves of common sensation produced pain, came to invade the neighbourhood of the nucleus of the vagus, it might be expected to give rise to symptoms like those of the gastric crises (pain in the stomach and vomiting), and I suggested this hypothesis. At the meeting of the International Medical Congress in London during the following year, Professor Pierret showed me sections of the medulla oblongata which he had recently made from a case of tabes with gastric crises. I was much interested in seeing that the structures in immediate relation with the nucleus of the vagus exhibited distinct sclerosis.

Since then, as far as I know, in every case of tabes with gastric or laryngeal crises of which a necropsy has been published (where the medulla oblongata has been examined), the presence of sclerosis in the neighbourhood of the vagal nuclei has been established. In a recent paper* Rosenthal says, "The anatomical basis for the cardialgia and hypersecretive vomiting may be found in demonstrable degenerations in the territory of the vagus centres." We may take it, indeed, I think, as determined that subsequent anatomical examination has justified the hypothesis brought forward by me ten years ago. It may also, I submit, be

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* *Wien. Med. Press*, 1887.

allowed that irritation of the nuclei of bulbar nerves will occasion symptoms indistinguishable from those which would be produced by irritation of the periphery of the nerve. The most important evidence bearing upon this point is derived from a case published by Dr. Sharkey,* in which the characteristic symptoms of Ménière's disease were due to the presence of an intra-cranial tumour lying against the pons varolii and involving the auditory nerve.

In the short space at my command on the present occasion I cannot adduce examples illustrative of this subject in any detail, but will refer briefly to a few.

A very hard-worked professional man, thirty-seven years old, began to suffer in September, 1888, from depression of spirits and tinnitus aurium, and went into the country to recruit. Whilst there he had an attack of vertigo, followed by vomiting and a feeling of great illness, from which after a few days he improved, but later on four occasions experienced a return of the symptoms at intervals of about a week. The attacks then left him, and when I saw the patient about six months afterwards there had been no further return. His hearing was then not quite satisfactory, and there was some tinnitus (noise of a fife) in the ears, especially the right one. During the attacks he had been (to use his own expression) nearly "stone deaf." I found that he could turn his head to either side without becoming giddy. This patient's father inherited and suffered severely from gout, and the patient himself three years previously had a slight attack of gout in the ball of one big toe, which lasted a week. He had been accustomed to eat a good deal of meat, to drink a fair amount of Australian wine, and to take but little exercise. Let me draw your attention to a point which appears important. During the attacks the patient said he had been nearly "stone deaf." It is clear that a deafness which is paroxysmal in this way cannot be dependent upon organic disease, and that we must look elsewhere than in the internal ear for an explanation of it.

A stout, well-nourished, temperate man, forty-six years of age, was taken with vertigo, relieved it was thought by brandy, but followed two hours later by a very violent return. This was coupled with vomiting, profuse sweating and violent palpitation. There was deafness and tinnitus in the

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* *Brain*, April, 1888.

left ear. When he attempted to sit up or turn towards the left side he became intensely giddy and felt extremely ill, vomiting would occur, and without support he would fall. He did not see things moving; the giddiness was "in himself." It was only on changing his position that he became uncomfortable. The first attack occurred half-an-hour after a light meal. Immediately before the attack he had been feeling in particularly good health. His appetite and digestion were good, and there had been no evidence of any stomach disorder. The inability to move without giddiness and vomiting lasted for about a fortnight, since which time, now a year and three-quarters, he has had no return. He has no deafness and no singing in the ear. The patient's pulse shows high tension, and there is a family history of gout. There is no evidence of cardiac disease. There was a history of palpitation of the heart and occasional faintness for ten or twelve years previously to the giddiness, with transient glycosuria. He had also suffered for some months from paralysis of one external rectus muscle.

A lady fifty years of age, stout, flabby, with strongly marked uric-acid diathesis, suffers from vomiting and retching irrespective of food being taken. There is no alcoholic excess. She will dream that she is being sick, and wake with an illusive sensation of vomit in her mouth. Along with this there is vertigo only on change of position —highly suggestive of Ménière's vertigo; but nothing whatever can be found wrong with her ears. She has no tinnitus and her hearing is perfect.

Cases of the kind are very numerous. You must all, as I have, have met with them very frequently. They seem to me, whilst manifestly possessing the characters of Ménière's vertigo, to point, not to any pathological change in the internal ear, but to a transient influence exerted upon the auditory nerve nucleus in the bulb. In such cases you will find that sometimes one nerve centre and sometimes another appears to bear the brunt of the influence. Violent attacks of vertigo with auditory symptoms may occur at irregular intervals, the patient enjoying good health meanwhile, and being quite free from symptoms pointing to disease of the ear. Perhaps a certain amount of tinnitus or deafness may remain, showing that the irritation of the centre has not entirely subsided. In other cases the patient may have an attack of irregularity of the

heart's action, comparable in its suddenness and violence with the vertigo, which it apparently replaces. Or the sensory nucleus of the fifth may be attacked, and the patient suffer from neuralgia as paroxysmal in character, occurring and subsiding as suddenly, as the vertigo with which he is seized on some other occasion. It appears to me that we can scarcely fail to see in the paroxysmal character of these various disorders, and their liability to occur in the same individual, evidence of a common causation affecting at different times various nerve centres in the bulb.

In case we are right in thinking that some irritative influence in the bulb is a very frequent determining agent in the production of giddiness and other troubles—vomiting, irregularity of the heart's action, neuralgia, glycosuria, faintness—is it possible to say what is the nature of this influence? The time has not yet arrived when anything definite can be said on this point. There appears, however, some reason to think that it may be due to the presence in the blood of uric-acid salt or some equivalent. Cases of the kind described are very numerous, and so common is it to find some gouty history in them that I constantly shape my inquiries and treatment in this direction, often, though not by any means always, with success.

It is customary to refer the cardiac symptoms to reflex irritation from a disordered stomach, but, if you inquire carefully into these cases, I think you will find, as I have, that there is usually no evidence whatever of a local gastric trouble. The tongue is clean, the appetite good, and the process of digestion is attended with no discomfort. In these circumstances it seems much more likely that the influence is exerted directly upon cardiac nerve centres in the bulb. The presence in the blood of some substance prone to show what has been termed "elective affinity" for certain structures would best explain the phenomena, and this substance may possibly be urate of soda, the presence of which in the blood of the gouty has been demonstrated by Garrod.

Many years since I brought forward clinical evidence which appeared to show that uric-acid salts by their presence in lymph spaces might produce symptoms of sciatica and infantile paralysis.* I would only now suggest that a

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* *The Practitioner*, 1877. *The Lancet*, 1880.

like source of irritation of nerve nuclei in the bulb is conceivable in connection with the lymph spaces in the walls of the blood vessels—but this is necessarily hypothetical.

It has long been my habit to prescribe salicylate of sodium in cases of vertigo associated with auditory nerve symptoms. The employment of this drug suggested itself partly on account of its influence in the elimination of uric acid, as pointed out some years ago by M. Germain Sée, and partly also because it apparently exerts some direct influence upon centres in the medulla oblongata, occasioning, as it does in full doses, reduction of temperature, headache, deafness, tinnitus aurium, giddiness, and sometimes dangerous depression of the heart's action. It is only, however, since I read the admirable papers of Dr. Alexander Haig on Uric Acid * that the advantage belonging to the employment of the salicylates has appeared to me to receive adequate explanation.

I have neither the time nor the ability to proceed further in a direction which would rapidly lead us into the question of the nature of gout, and its possible connection with a nervous origin—a question which has been so ably discussed by Sir Dyce Duckworth. The object of this address has been to urge that we must often look to something in the bulb itself for the explanation of vertigo with auditory symptoms and allied disorders of function in the district of bulbar nerves. What that something is I cannot pretend to say. It may be uric-acid salt, or possibly some other product of disturbed metabolism.

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* "Medico-Chirurgical Transactions," 1887. "The Formation and Excretion of Uric Acid," 1888.